DOCUMENT RESUME

ED 318 140 EC 230 091

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TITLE Intellectual Characteristics of Diabetic Children at Diagnosis and One Year Later.
INSTITUTION Hospital for Sick Children, Toronto (Ontario).
PUB DATE 89
NOTE 24p.
PUB TYPE Reports - Research/Technical (143)

EDRS PRICE MF01/PC01 Plus Postage.
DESCRIPTORS Children; *Cognitive Ability; Correlation; *Diabetes; Foreign Countries; *Intelligence Quotient; Longitudinal Studies; Spatial Ability; Verbal Ability; Young Children

ABSTRACT

A longitudinal investigation of neurocognitive function was conducted on 63 newly diagnosed pediatric patients with insulin-dependent diabetes mellitus (IDDM) at onset of illness and 1-year post diagnosis. The results at diagnosis, when the children were ages 4 months to 12 years, revealed no differences compared to sibling controls, nor any specific impairment predating illness. No evidence of any acquired impairment was found following 1 year of diabetes, although children who developed IDDM before age 5 performed more poorly than those with later onset on spatial tasks, while those with later onset scored lower on verbal tasks. Episodes of asymptomatic and mild chronic hypoglycemia were positively correlated with improved outcome over time. There were no adverse effects of severe hypoglycemia. Ketonuria and hospitalizations were associated with lower performance intelligence quotients 1 year post diagnosis, as was diabetic ketoacidosis at onset. The results are discussed in terms of critical periods of sensitivity of the brain to diabetes and the need for longer follow-up of these children. Includes 53 references. (JDD)

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INTELLECTUAL CHARACTERISTICS OF DIABETIC CHILDREN AT DIAGNOSIS AND ONE YEAR LATER

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Keywords. Diabetes, children, neuropsychology, hypoglycemia

EC 230091

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A longitudinal investigation of neurocognitive function was conducted on 65 newly diagnosed pediatric patients with insulin-dependent diabetes mellitus (IDDM) at onset of illness and one-year post diagnosis. The results at diagnosis revealed no differences from sibling controls nor any specific impairment predating illness. No evidence of any acquired impairment was found following one year of diabetes, although children who developed IDDM before age 5 performed more poorly than those with later onset on spatial tasks while those with later onset scored lower on verbal tasks. Episodes of asymptomatic and mild chronic hypoglycemia were positively, not negatively, correlated with improved outcome over time. There were no adverse effects of severe hypoglycemia. Ketonuria and hospitalizations were associated with lower PIQs one year post diagnosis, as was DKA at onset. The results are discussed in terms of critical periods of sensitivity of the brain to diabetes and the need for longer follow-up of these children.

INTELLECTUAL CHARACTERISTICS OF DIABETIC CHILDREN AT DIAGNOSIS AND ONE YEAR LATER

Insulin- dependent diabetes mellitus (IDDM) is one of the commonest chronic childhood diseases, occurring in about 1 in every 600 children under the age of 12 (Traisman 1980). It is caused by a pancreatic disorder that results in a lack of insulin production, insulin being an essential hormone for glucose metabolism. Without insulin, glucose accumulates in the bloodstream and urine while the body draws on other sources of energy until it literally starves. If untreated, severe hyperglycemia (high blood sugar) can lead to ketoacidosis, diabetic coma, and ultimately, death. Since the discovery of exogenous insulin in 1922 (Banting, 1929), persons with diabetes approximate normal lives by taking daily insulin injections, the dose of which is regulated with exercise, diet and stressful events, including illness. If properly controlled, most individuals with diabetes will have few consequences of diabetes until at least mid-adulthood when complications such as neuropathy, retinopathy, and nephropathy, resulting from long-standing chronic hyperglycemia, may develop (Travis, Brounard, Schreiner, 1987; Drash, 1986). In order to prevent these later complications, diabetologists require strict control and adherence along with close monitoring of blood glucose levels. However, for individuals with diabetes, it is not simply sufficient to prevent hyperglycemia, it is also necessary to restrict hypoglycemia (low blood sugar) which occurs from too much insulin, too little food, or an imbalance of both with exercise and/or emotional state. Severe hyoglycemia can cause seizures or coma while moderate hypoglycemia may affect cognition (Holmes, Hayford, Gonzalez, Weydert, 1983) and emotional state (Gonder-Fredrick,1989). Maintaining a tight balance between hypo- and hyperglycemic states is not an easy task, particularly with pediatric patients. According to Berg and Linton (1988) "this metabolic seesaw may have important implications not only for the psychological adjustment, but also for the brain of the diabetic youngster" (p.113).

Despite the burden of diabetes and the difficulties for all family members, in adjusting to this chronic illness, diabetes has traditionally been thought to be relatively benign with respect to intellectual status and functioning. Based on their clinical impressions and anecdotal evidence, many diebetologists believed that not only were these children of normal intelligence, they were likely to be above average in this regard (Brown & Thompson, 1937). However in 1961, Ack, Miller and Weil reported that among diabetic children, those who were diagnosed before age 5 had significantly lower IQs than sibling controls and others with diabetes. Because the early onset group did not differ from controls in their school performance (Weil & Ack, 1964), this difference was not considered to be clinically significant. For the next 20 years most research on diabetic children focussed on the psychosocial aspects of this disease (e.g. Barglow, Edidon, Budlong-Spring,1983; Gath, Smith & Baum, 1980; Gross, Delcher, Snitzer, Bianchi, Epstein, 1986; Mazze, Lucido, Shamoon, 1984; Swift Seidman, Stein,1967; see Bennett-Johnston, 1980 for a review), even though a small number of electrophysiologic studies reported abnormalities in CNS function among diabetic children and adolescents (Eeg-Olofsen & Petersen, 1966; Gilhaus, Daweke, Lulsdorf, Sachsse, Sachsse, 1973; Haumont, Dorchy, Pelc,1979)

In recent years, five teams of investigators have re-investigated the issue regarding neurobehavioural status in these pediatric patients (Anderson, Hagen, Barclay, Goldstein, Kandt, Bacon, 1984; Hale, Berenbaum, Traisman, Golden, Rosenberg, Headen, 1985; Holmes & Richman, 1985; Rovet, Ehrlich & Hoppe, 1987, 1988; Ryan, Vega, Drash, 1985). They observed that while diabetic children and adolescents may be unaffected on average, certain subgroups appear to be at increased risk for subsequent neurocognitive impairment. All five studies reported that children diagnosed before the age of five typically performed more poorly on tests of visuospatial and other aspects of neurocognitive functioning than controls or children who developed diabetes later. Several investigators have in

addition attributed these deficits to moderate and/or severe hypoglycemia occurring early in childhood (Ryan et al,1984; Rovet et al, 1987).

Because this research was cross-sectional and retrospective in design, there was no way of knowing whether the deficits preceded illness or were acquired as a result of it. It was also not possible to determine precisely the contributing diabetes-related factors, because one had to rely primarily on parental recall of previous glycemic events. In order to examine the neurocognitive characteristics of diabetic patients in greater detail, we have been conducting a prospective investigation on an unselected population involving a large consecutive cohort of newly diagnosed patients who have been followed through the first three years of their illness. Our goals were to determine whether there are deficits at the time of onset, the changes that may occur in performance at the end of one and three years of illness, the factors contributing to any such changes by recording glycemic episodes on an ongoing basis throughout the three year interval, and the children at greatest risk for subsequent impairment. This paper reports our findings at diagnosis and after one year of diabetes.

METHODS

Subjects

The sample consisted of 63 children (33 boys, 30 girls) with newly diagnosed diabetes mellitus, 23 children of whom were under 5 at the time of diagnosis. The mean (SD) age was 7.32 (4.3) years (range = 0.4 to 12.5 years). The children represented 72% of all eligible cases at the Hospital for Sick Children between April 1984 and February 1987 with newly diagnosed IDDM. Criteria for inclusion were (i) age below 12 years; (ii) English-speaking; (iii) residing in the immediate Toronto vicinity; (iv) no other major illnesses or disabilities; and (v) parental agreement. Controls consisted of 40 non-diabetic siblings under the age of 12 years (mean age =8.17 (3.8) years).

Families were initially approached during their child's initial hospital admission with diabetes for participation in this study. Following consent, an appointment was scheduled for an assessment within the next two months, preferably the next month. Children with diabetes were assessed at onset and annually thereafter for the next 3 years. Families were interviewed every three months during the child's diabetes clinic visit. as to recent diabetic history. Controls were seen along with the diabetic child at his or her initial and final (3 years) visits. A parent, usually mother, was also assessed for intelligence at the initial visit.

This study was approved by the Hospital's Human Subjects Committee.

Test Measures

Neurocognitive. All subjects were evaluated with a battery of tasks that included measures of general intelligence, visuospatial abilities, language, and when applicable, memory and achievement. Because of the large age range of the sample, children at different ages had to be assessed with different test instruments. These are listed in Table I.

Insert Table I about here

The Griffiths provides a Developmental Quotient (DQ) and Standard Scores on up to 6 scales of neurocognitive functioning: (A) Locomotor, (B) Personal Social, (C) Hearing and Speech, (D) Eye-hand Coordination, (E) Perceptual Performance, (F) Verbal Reasoning. For comparison of scores with the Wechsler scales given to older children, the mean of C and F was used as a measure of VIQ and the mean of D and E, as a measure of PIQ.

Parental intelligence was determined using an abbreviated WAIS-R (Wechsler, 1979); socioeconomic status of the family with the Hollingshead 4-Factor Index (Hollingshead & Redlich, 1964).

Diabetic Information. The PH level at diagnosis was recorded with values below 7.3 being considered acidotic (Travis et al, 1987). At every

trimonthly diabetes clinic visit, the child's diabetic diary was copied and the family was questioned as to its contents and specific diabetes-related events. The following information was also obtained: # monthly low blood sugar readings below 4.0 mM/dl (72 mg/L) without symptoms; # per month, symptomatic low blood sugar recordings (dizziness, sweaty, shaky); #severe low blood sugar episodes (unconsciousness, convulsions); # monthly high blood sugar readings (>13.3 mM/dl) with ketones present in urine (ketonuria); #hospitalizations with and without diabetic ketoacidosis (DKA). In addition each child's HBA1c value, obtained trimonthly, was also recorded.

RESULTS

The mean (SD) parental IQ was 103.7 (15.5), which is within normal limits. Subjects spanned the full SES range (I: 19%; II: 27%; III: 19%; IV: 15%; V: 19%), with a slight over-representation at the higher end. Ninty-six percent were white, 2% were black, 0% oriental and 2% east Indian. This does not reflect Toronto's large multi-racial community, including its large oriental population. There were no differences between children with early and late onset on any demographic variables.

Table II provides the results for diabetic children and sibling controls. At the time of diagnosis, diabetic children scored signficantly higher on the WISC-R Similarities subtest ($t=2.02$, $p<.05$) and showed a trend towards higher scores on Arithmetic ($p<.09$) and Picture Arrangement ($p<.07$) than controls. The groups did not differ on any measure of achievement or spatial ability. Table II also shows no evidence of any specific impairment in the diabetic children at the time of diagnosis of disease as reflected by the pattern of subtest scores, which was confirmed by no reports from the mothers of any specific learning impairment.

Insert Table II about here

Sixty diabetic children were available for retest one year post-diagnosis. The mean scores for these children after one year of diabetes are also given in Table II. Matched t-test comparisons indicated improved performance on the following indices: Griffith Personal Social, $t = 1.74$, $p<.10$, Griffiths Hearing and Speech, $t = 3.16$, $p<.01$, Reynell Comprehension, $t=1.74$, $p<.10$, WISC-R Digit Span, $t=3.34$, $p<.01$, WISC-R Object Assembly, $t=1.75$, $p<.10$, WRAT Reading $t=1.99$, $p<.05$, and Woodcock-Johnson Spatial Relations, $t=2.74$, p.01. The only index involving a decline was the WISC-R Vocabulary subtest, $t=2.08$, $p<.05$, although actual performance still remained in the average range. These results indicate that while the younger children, who were tested with the Griffiths and Reynell Language Scales, showed improved verbal skills after one year, older children, tested with the WISC-R, showed a decline in verbal functions.

Unlike the retrospective studies where children with early and late onset were the same age at time of testing but differed in duration of illness, the children in this study had the same length of illness but, of course, differed widely in the age at testing, which necessitated the use of different test instruments. This meant they could not be directly compared on individual subtests, only summary IQ scores. Furthermore, because of the age restrictions of the tests, the younger i.e. early onset, children could not be tested with the more sensitive cognitive processing measures, especially in the area of spatial abilities. The results for the 23 early onset and 40 later onset children are provided in Table III. Comparisons of these groups revealed that children with early onset showed a tendency for lower PIQ values at onset than did those with later onset ($p<.06$), which was sustained over the one year interval ($p<.09$). Children with later onset, by contrast, obtained slightly lower VIQ scores at onset with this differential increasing substantially ($t=2.49$, $p<.01$) after one year of diabetes.

Insert Table III about here

Table IV provides the information on the diabetes-related variables over the first year period. The incidence of severe reactions was extremely low with only 2 children experiencing a hypoglycemic convulsion and only 1 an episode of DKA following diagnosis. Correlations of diabetes data with outcome at one year were significant only for number of recorded hypoglycemic reactions and performance in the verbal area of cognitive functioning. However, the direction of the correlation was consistently positive, reflecting the fact that children with more recorded reactions had higher IQ ($p<.01$), VIQ ($p<.01$), Information ($p<.01$), Vocabulary ($p<.01$), Similarities ($p<.01$), Digit Span ($p<.01$), Reading ($p<.01$), Spelling ($p<.01$) scores. Results on spatial tasks were not correlated with any indices of diabetic control.

Insert Table IV about here

Each child with diabetes was also assigned a verbal and a visuoperceptual change score using the following procedure: VIQ (or PIQ) scores at one year were regressed on VIQ (or PIQ) scores at onset and the residuals of each child's pair of scores to the regression lines were computed. To account for the fact that children may have been given different test combinations at the two intervals, and because the relationships between tests could have differed for different test combinations during this period, separate series of regressions were computed for children receiving the Griffiths at both intervals, the Griffiths and then the WPPSI, the WPPSI and then the WISCR and the WISCR at both intervals. The change scores for the entire cohort were combined and correlated with diabetes-related indices.

Hypoglycemia (asymptomatic, mild, moderate and severe) was not associated with poorer skills in either verbal or spatial domains. Instead, we observed a tendency for *more* asymptomatic low blood sugar readings at the third and fourth trimonthly intervals and more mild to moderate hypoglycemic reactions over the first three months to be associated with *improved* verbal ability one year post diagnosis ($p<.10$). By contrast, poorer outcome in the perceptual domain was correlated with more episodes of recorded ketonuria at the first ($p<.01$) and third ($p<.10$) quartiles. Hospitalizations during the first year were asssociated with larger declines in PIQ ($p<.05$).

The final analysis concerned the effect of diabetic ketoacidosis (DKA) at onset on outcome. Children were grouped by their PH levels at onset of diabetes, with those having a PH value below 7.3 being considered as having DKA and those with a PH value above this level, no DKA. Comparisons of these two groups revealed no difference on any index at onset of diabetes. However after one year of illness, the DKA group showed a tendency towards lower scores than the no DKA group on WPPSI Mazes (DKA=9.7, no DKA = 13.4; $t=-2.29$, $p<.06$) and WISC-R Object Assembly (DKA=11.1, no DKA=13.0, $t=-1.93$, $p<.06$). Although not significant, children in the DKA group also had slightly lower IQs at T1 (113.5 versus 116.2).

DISCUSSION

Present findings suggest no evidence of any neurocognitive impairment in diabetic children at the time of onset of disease or after one year of illness. If anything, some skills appeared to improve with time, possibly because the children were feeling better physically or had learned to adapt to and cope with the disease (Kovacs, Brent, Steinberg, Paulauskas, Reid, 1986; Kovacs, Feinberg, Paulauskas, Finkelstein, Pollock; Novak, 1985). Children with early disease onset showed no evidence of any spatial impairment, although children with later disease onset indicated a minor decline in their Vocabulary skills. Preliminary findings from those children

completing the three year phase of the project, show that the effects in the verbal domain tend to persist while differences in the spatial begin to emerge (Rovet, 1989).

There were few negative effects on neurocognitive outcome associated with the different diabetic control factors. One index, number of mild-moderate hypoglycemic symptoms recorded in the diaries, in fact, was positively associated with better outcome in the verbal area of cognitive functioning. Although this most likely was artifactual since mothers of children with improved functioning may have been more assiduous in recording events in their children's diaries, it could have been an indirect reflection of tighter control over hyperglycemia. Episodes of more severe manifestions of diabetes (severe hypoglycemia, DKA after onset) were extremely rare in the present sample, presumably because the children were probably still in the honeymoon phase of the disease (i.e. they were most likely still experiencing some residual endogenous insulin secretion). Ketonuria and hospitalizations were negatively correlated with outcome in the nonverbal visuospatial domain.

There are several explanations for the difference between present findings and those observed in previous retrospective studies with diabetic children. First, the lack of a spatial deficit in the early onset children could be that deficits have occurred but they are latent as the children are too young to be tested for such or have not as yet encountered the requisite spatial skills. Even though prepubertal duration of diabetes appears to be a very weak predictor of outcome for other diabetic complications (Norris et al, 1989), it may be that in the neuropsychological domain, the effects of chronic diabetes are cumulative and take more time than one or even three years (Rovet, 1989) to appear. This suggests that the sample should be followed for longer periods of time before final conclusions are drawn. These arguments have assumed that the retropsective studies with their highly consistent findings are correct and present findings are at variance.

Alternatively, it may be that children in the present sample will not be impaired, because they have been drawn from a different patient population than the children in the retrospective studies, who were grew up in the era preceding self-monitored blood glucose recording, glucometers and generally tighter control (Skyler, 1989). It is, therefore, extremely important to follow the children in our study over a longer time frame in order to see if present treatment methodologies do signify better outcome in recently diagnosed diabetic children.

The observation that older onset diabetic children may decline in their verbal skills has not been explicitly reported in previous work, although Ryan et al (1984) did observe that verbal conceptual skills were lower for adolescents tested with the WAIS (>16 years) than those tested with the WISC-R (<16 years). They attributed this difference, however, to the cumulative effects of more frequent school absence in older children rather than age at onset or at testing effects per se (Ryan, Longstreet, Morrow, 1985). Nevertheless it is possible that this observation actually represents *a second age of onset effect* reflecting what happens in the brain when diabetes presents at a later age. We have recently proposed that there may be different critical periods of sensitivity of different functional and/or structural substrates in the brain (Lauder & Krebs, 1986; Greenough, 1986) to the effects of diabetes such that those underlying spatial abilities are more vulnerable to diabetes presenting at an earlier age and those underlying verbal abilities being more vulnerable to diabetes presenting later (Rovet, Ehrlich, Czuchta, 1989). We have speculated that one possible mechanism contributing to these specific deficits is the disruptive effect of chronic hyperglycemia on myelin formation (Rovet, 1989). This follows from Yakolev and Lacours (1964) histopathological studies snowing that different cerebral structures appear to myelinate at different rates and times during the first (or later) decade of life. In support of this is the recent study by Vlassara, Brownlee, and Cerami

(1983) in which they showed that myelin formation was altered within a relatively short period of rats being made experimentally diabetic.

Another factor contributing to specific neurocognitive impairment may be the disruptive effects of diabetic ketoasidosis (DKA) that often accompanies the onset of illness. Tsalikian, Daneman, Becker, Crumrine, Drash (1980) reported an increased incidence of abnormal EEGs in children following DKA at onset, which did not resolve in a significant number of the children with time. After one year we found two aspects of cognitive functioning, planning skills and part-whole perception to be slightly weaker in the group who experienced DKA. In addition, among the children who have now had diabetes for three years, our preliminary findings indicate that a larger number of neurocognitive functions appear to be subsequently disrupted by DKA at onset i.e. 3 years earlier, with different types of impairment reflecting age at time of the episode (Rovet, 1989). Among children less than five years of age at diagnosis, those with DKA scored lower on a number of verbal and spatial measures while children between five and nine years showed no effect and children above 9 at the time of DKA at onset did more poorly in verbal, numerical and memory abilities. These results suggest that it may take time for the effects of DKA to be expressed and that the effects vary for different age groups, who presumably differ in stage of brain maturation and the particular vulnerability of different cerebral substrates at the time of insult.

In conclusion while present findings suggest generally good prognosis for diabetic children in terms of neurocognitive outcome, it may be premature to draw definitive conclusions until longer term effects have been observed. Present findings, as well as preliminary results on three year data (Rovet, 1989), do not indicate any negative impact of mild or severe hypoglycemia. However, DKA with onset and frequent hyperglycemia may adversely affect specific aspects of neurocognitive functioning.

ACKNOWLEDGEMENTS

This work has been supported by research grants from the Canadian Diabetes Association and a Scholarship from Health and Welfare Canada. Portions of this work have been presented at the Third Invitational Conference on Behavioural Diabetes, Newport Beach, February 1989, the Society for Research in Child Development, Kansas City, April 1989, and as part of the invited symposium, The Neuropsychological Sequelae of Diabetes, at the American Diabetes Association meeting, Detroit, June 1989. The authors are especially grateful to Jeannine Pinsonneault and Donna Sorbara for their contributions in carrying out the study, to Leslie Langdon and Cathy Jackson for the initial assessments, to Joan Ronde for secretarial support, and to Gary Ingersoll for his suggestions in deriving the change scores.

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Table I. Test Battery

Ability	Test Measure	Age Given
General Intelligence	Griffiths (Griffiths, 1967)	<4
	WPPSI (Wechsler, 1970)	4-5.9
	WISC-R (Wechsler, 1974)	>6
Verbal Abilities	Reynell (Reynell 1978)	<4
	Griffiths C	
	WPPSI, WISC-R verbal subtests	>4
Spatial Ability	Griffiths E	>4
	WPSSI, WISC-R subtests	>4
	Test of Visuomotor Integration (Beery & Buktenica, 1967)	>4
	PMA Spatial (Thurstone, 1962)	>6
	Woodcock Johnston Spatial Relations (Woodcock Johnson 1979)	>6
Memory	WPPSI Sentences	4-6
	WISC-R Digit Span	>6
Academic Achievement	Wide Range Achievement Test (Jastak & Wilkinson, 1978)	>6

Table II. Performance by Diabetic Children and Siblings at Diagnosis (T_0) and after One Year Diabetes (T_1)

	T_0				T_1	
	Diabetic (N=63)		Sibling (N=40)		Diabetic (N=60)	
Test	M	SD	M	SD	M	SD
IQ						
Full Scale	113.3	13.1	110.8	12.9	115.7	13.4
Verbal	111.0	16.8	108.1	16.5	113.2	16.2
Performance	113.4	11.6	116.2	13.3	111.9	14.0
Griffiths[a]						
A. Locomotor	110.6	8.8	112.2	24.5	114.8	11.4
B. Personal Social	112.0	12.3	108.2	19.5	119.8	11.8
C. Hearing & Speech	111.6	23.5	114.0	23.4	126.0	21.4
D. Eye-Hand	108.1	10.5	112.6	15.4	105.5	17.8
E. Performance	110.0	14.8	113.2	34.0	112.6	11.0
F. Reasoning	116.2	14.4	109.5	44.5	114.3	15.3
Reynell[a]						
Expressive	0.294	1.35	0.450	1.34	0.667	1.05
Comprehension	0.413	1.23	0.100	1.84	1.200	0.94
WISC-R or WPPSI						
Information[b]	10.8	3.4	11.4	3.0	11.3	3.1
Vocabulary[b,j]	12.1	2.9	11.5	3.4	11.6	3.0
Arithmetic[b]	11.4	3.1	10.6	2.6	11.2	2.8
Similarities[b,i]	12.8	3.5	11.1	3.8	12.9	3.2
Comprehension[b]	12.4	3.0	12.8	2.3	13.4	3.0
Picture Compl.[b]	12.2	2.5	13.2	2.7	12.1	2.1
Block Design[b]	12.9	2.7	12.5	2.8	12.0	2.6
Sentences[c]	11.0	2.8	12.5	5.0	9.8	2.5
Animal House[c]	13.3	2.0	12.0	1.6	13.4	3.0
Mazes[c]	12.0	4.3	12.0	2.9	11.2	2.5
Geometric Design[c]	13.3	2.1	12.0	5.0	14.0	5.0
Digit Span[d,h]	8.7	2.8	10.4	2.8	10.1	3.2
Picture Arrang.[d]	12.8	2.9	12.4	2.5	10.9	2.7
Obj. Assembly[d]	12.0	3.1	12.5	2.9	11.9	2.9
Coding[d]	11.7	2.8	12.2	3.2	10.8	3.2

	T_0				T_1	
	Diabetic (N=63)		Sibling (N=40)		Diabetic (N=60)	
Test	M	SD	M	SD	M	SD
WRAT-R (%ile)[e]						
Reading[h]	48.8	27.7	53.3	28.2	40.3	31.5
Spelling	45.2	29.5	49.2	24.3	37.0	30.5
Arithmetic	36.6	28.5	43.0	27.9	38.5	27.9
Beery (T-Score)[f]	63.1	26.9	62.0	26.5	60.3	26.1
Woodcock Spatial						
Rel. (%ile)[g,h]	61.6	24.9	72.6	19.6	48.2	23.8
PMA Spatial (SS)[g]	114.0	16.5	116.3	18.8	112.2	15.4

[a]Based on 20 Diabetics and 5 Siblings at T_0, 12 Diabetics at T_1
[b]Based on 43 Diabetics and 35 Siblings at T_0, 48 Diabetics at T_1
[c]Based on 6 Diabetics and 6 Siblings at T_0, 8 Diabetics at T_1
[d]Based on 37 Diabetics and 28 Siblings at T_0, 40 Diabetics at T_1
[e]Based on 40 Diabetics and 33 Siblings at T_0, 40 Diabetics at T_1
[f]Based on 57 Diabetics and 35 Siblings at T_0, 52 Diabetics at T_1
[g]Based on 38 Diabetics and 30 Siblings at T_0, 39 Diabetics at T_1
[h]$T_1 > T_0$, $p < 0.01$
[i]Diabetic > Sibling, $p < 0.05$
[j]$T_0 > T_1$, $p < 0.07$

Table III Comparison of Early Onset Diabetic (EOD) and Later Onset Diabetic (LOD) Children and Diagnosis (T_0) and after One Year (T_1)

	T_0		T_1	
	EOD (N=23)	LOD (N=40)	EOD (N=21)	LOD (N=39)
IQ	112.8	113.7	117.4	114.8
VIQ	114.5	109.0	119.9	109.5[a]
PIQ	109.8	115.4[b]	112.2	118.3[b]

a = $p < 0.01$
b = $p < 0.10$

Table IV Diabetes-Related Information

% with PH <7.30 at diagnosis	32.2%
PH at diagnosis	7.27 ± 0.64
HBAIc	
1st 3 months	8.29 ± 1.52
2nd 3 months	8.64 ± 3.03
34d 3 months	8.52 ± 1.45
4th 3 months	8.67 ± 1.36
Asymptomatic Low Blood Sugars/month	3.58 ± 3.28
Reactions/month	0.46 ± 0.96
Ketonuria/month	0.57 ± 1.30
% having convulsions	3.2%
% having DKA (after onset)	1.5%
Hospitalizations/subject/year	0.126